

patient who had primary mediastinal choriocarcinoma with widespread lung metastasis and hyperthyroidism. Case: 18-year-old male presented to the emergency department for a fever of 105 F, cough, shortness of breath and night sweats for 9 days. He also reported unintentional weight loss for the past few weeks. Upon presentation, he was tachycardic at 104 beats/min, tachypneic and febrile. Physical examination revealed bibasilar crackles. Thyroid and scrotal examination were unremarkable. Laboratory work was significant for leukocytosis of 18.7 k/cmm, AST: 69 Unit/L, ALT: 60 Unit/L, ALP: 123 unit/L and LDH: 438Unit/L. Chest radiology showed bilateral multiple cannonball lesions with mediastinal and periaortic lymphadenopathy. CT chest, abdomen, and pelvis revealed innumerable diffuse bilateral lung masses, large anterior mediastinal mass, enlarged periaortic and paratracheal lymph nodes. Infectious workup was unremarkable. MRI brain revealed leptomeningeal involvement and vasogenic edema. Ultrasound of testicles revealed bilateral severe microlithiasis. CT guided biopsy of lung mass showed poorly differentiated choriocarcinoma. Treatment was initiated with systemic steroids, bleomycin, and cisplatin. Additional blood work revealed, B-HCG:736,598 mIU/mL, TSH:0.009UIU/ml, free T4:2.95ng/dL and total T3: 292.3ng/Dl. Thyroid radioiodine uptake scan was normal and planar imaging was also normal. Treatment with methimazole was deferred due to the up-trending transaminases and the resolution of tachycardia. Repeat labs after chemotherapy were TSH: 0.005UIU/mL, free T4: 2.04ng/dL, free T3:1.70pg/mL and BHCG: 202,471UIU/mL. Unfortunately, the patient had chemotherapy-resistant choriocarcinoma and succumbed to death after 4 cycles. Discussion: Beta HCG has an intrinsic thyroid-stimulating activity. The HCG -alpha subunit is common to all glycoprotein hormones like follicle-stimulating hormone, luteinizing hormone or thyroid-stimulating hormone. Frankly elevated levels should be treated symptomatically with beta-blockers and/or anti-thyroid drugs. Subclinical hyperthyroidism should be monitored closely. Some patients do not develop hyperthyroidism or a toxic goiter, as it is dependent on the timing of onset of cancer or due to the secondary modifications of HCG that can affect the bioactivity of HCG on the TSH receptor. This case draws our attention to the importance of focusing on the molecular mimicry of the HCG and TSH and management of HCG mediated hyperthyroidism.

Thyroid

THYROID DISORDERS CASE REPORTS II

Amiodarone Induced Myxedema Coma

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SAT-LB83

Case Report: Amiodarone Induced Myxedema Coma Sabah Syed, Keun Young Kim

Introduction Amiodarone is widely used as a rhythm-conversion agent in atrial fibrillation. It's low negative inotropic activity and low risk of ventricular arrhythmia makes it an advantageous agent for atrial arrhythmia. One of the major limiting factors in the use of amiodarone can be

its thyroid toxicity leading to thyroid-dysfunction; ranging from thyrotoxicosis to myxedema coma. Myxedema coma, although rare, is a deadly complication of hypothyroidism with a mortality rate as high as 60%. It is considered an endocrinologic emergency that requires prompt attention and treatment. We present a case of myxedema coma in the setting of chronic amiodarone intake.

Case Presentation A 75-year-old female with a past medical history significant for atrial fibrillation, hypothyroidism, end stage renal disease from diabetic nephropathy on hemodialysis, Hepatitis C, hypertension, and hyperlipidemia was admitted to the hospital for evaluation of generalized weakness. Her medications included amiodarone 200mg/day among others. The next day, the rapid response team was called when the patient became hypotensive, bradycardic and unresponsive. She was transferred to the ICU and intubated immediately. On exam, her skin was dry and cold. Her laboratory findings revealed a markedly elevated TSH level of 185uIU/mL. Her most recent TSH level was 9.044uIU/mL from a year back. Other laboratory tests revealed WBC count of 9.0k/mm cu, hemoglobin 13.7 g/dL, platelet count 48 k/mm cu. A diagnosis of myxedema coma was made based on her presentation and lab findings. The patient was started on intravenous levothyroxine 100mg daily with intravenous hydrocortisone 100mg every 8 hours. T4 levels were 0.38ng/dL, T3 levels were 1.83pg/mL and TPO antibodies were negative. A decision was made not to administer intravenous T3 because of the patient's advanced age and underlying atrial fibrillation. Over the course of the hospitalization, the patient was showing signs of clinical improvement and attempts were being made to wean her off the ventilator. However, later she died due to hemorrhagic shock.

Conclusion Myxedema coma may manifest nonspecifically as lethargy and altered mental status and can present without the more specific symptoms of skin changes or myxedematous soft tissue changes. It is therefore, very important for physicians to consider myxedema coma as one of the differential diagnoses in patients on amiodarone with underlying thyroid disease. Administering amiodarone for elderly patients with underlying thyroid problems warrants attention to polypharmacy. The treatment guidelines for myxedema coma are yet to be established. While intravenous levothyroxine with intravenous hydrocortisone remains the mainstay of therapy, there are no consensus on T3 administration.

Bone and Mineral Metabolism

BONE AND MINERAL CASE REPORTS II

Hypocalcemia-A Clue to Digeorge Syndrome; A Very Rare Case Diagnosed in a 41 Year Male

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MON-LB63

Hypocalcemia- a clue to DiGeorge syndrome; a very rare case diagnosed in a 41 year male.

41-year-old male with history of anxiety, bipolar disorder, depression, cognitive developmental delay, Idiopathic Thrombocytopenic Purpura (ITP), Vit D deficiency, tinnitus, suicidal attempt, auditory hallucination, borderline intellectual functioning comes to the PCP's office for regular follow-up. The PCP has noted that the patient was hypocalcemic on multiple visits. On exam, vitals were in normal range, height 5 feet 2 inches, BMI 31 kg/m². Despite the patient being on calcium and Vit D2 pills for a couple of months, his Calcium level were low ranging from 5.8–7.8mg/dl (normal 8.5-10.1) with normal albumin; ionized calcium was low 0.9-0.97mmol/l (normal 1.12-1.23); Magnesium was normal 2mEq/L (normal 1.5-2.5), phosphorus slightly high 5 mg/dL (normal 2.5-4.9), PTH (ParaThyroid Hormone level) was low-normal 18.8pg/ml (normal 11.1-79.5), vitamin D 25 OH low 16ng/ml (normal 30-100); 1,25 di OH vitamin D low 8ng/ml (normal 25-40). Thyroid ultrasound showed 2 benign nodules, no further work-up done. TSH and free T4 were normal. FISH (Fluorescence In Situ Hybridization) came positive for DiGeorge syndrome (deletion at 22q11.2). He was treated with calcium 600 mg 4 pills daily, calcitriol 0.25 mcg two pills daily, Vit D2 50000 IU weekly and thiazide diuretic. His labs improved.

DISCUSSION Hypocalcemia can be due to low magnesium level, drugs or associated with high or low PTH. The patient had normal magnesium and he was not on any medication that causes hypocalcemia. This rules out first two cases. Hypocalcemia with high PTH (pseudohypoparathyroidism or low vit D levels) doesn't fall in our differential because our patient had low PTH. For hypocalcemia with low PTH, differentials include post-surgical condition (no surgical history), autoimmune (history and labs not suggestive of), infiltrative diseases (eg hemochromatosis- he had normal iron study, Wilson disease- normal copper level, granulomas- contrary he had low 1,25 Di OH vit D). Hypocalcemia secondary to genetic parathyroid gland anomaly was thought to be most likely in our patient. So FISH was pursued.

Conclusion: For patients with cognitive issues, persistence of chronic hypocalcemia (with low PTH) despite treatment should prompt for genetic disorders like DiGeorge. DiGeorge is usually the diagnosis of children. Perhaps this is the first case of DiGeorge diagnosed so late at age of 41.

Thyroid

THYROID DISORDERS CASE REPORTS II

Improvement In Metabolic Indices Including Thyroid Hormones Via Enhanced Absorption Of Nutrients By Teduglutide In Short Bowel Syndrome

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SAT-LB86

Background: Short bowel syndrome is characterized by maldigestion and malabsorption resulting in deficiencies of multiple nutrients including vitamins and minerals. Most subjects required parental elimination for survival. GLP 2 RA Teduglutide was recently approved for treatment of short-bowel syndrome especially for those requiring parenteral support. **Objective:** To demonstrate the utility of GLP2 Receptor Agonist Teduglutide in improving

multiple metabolic indices In presence of short bowel syndrome. **Case presentation:** 66-year-old Caucasian female presented with a history of short bowel syndrome and associated vitamin deficiencies, hypothyroidism requiring large dose of levothyroxine, diarrhea, and liver cirrhosis. Upon starting teduglutide the subject saw improvement in her symptoms. Moreover, daily dose of levothyroxine required reductions from 300 mcg to 150 mcg to maintain desirable serum concentrations of free T4, free T3 and TSH. Finally, serum levels of several vitamins attained greater than therapeutic concentrations requiring dosage reductions. Also notable was the improvement in her liver function tests, remission from ascites and regeneration of liver nodules. **Conclusion:** Herein, we report an adult subject with short bowel syndrome with concurrent hypothyroidism and multiple vitamin deficiencies who following administration of GLP2 RA therapy demonstrated a marked improvement in her metabolic parameters with some requiring reduction in daily dose along with improvement in manifestations of liver cirrhosis.

Pediatric Endocrinology

PEDIATRIC ENDOCRINE CASE REPORTS II

Retrospective Comparison of Cystic Fibrosis Related Diabetes Pediatric Screening Rates

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MON-LB011

Cystic fibrosis-related diabetes (CFRD) is the most common comorbidity in those with CF, affecting 20% of adolescents and 40-50% of adults with CF. If uncontrolled, it can cause worsened pulmonary outcomes, increased hospital length of stay, and increased mortality. It is typically clinically silent, and hemoglobin A1C and fasting plasma glucose are not sensitive enough to diagnose it. Per national guidelines, the proper outpatient screening method is oral glucose tolerance test (OGTT), annually beginning age 10. Inpatient diagnosis involves fasting glucose >126 mg/dl or 2 hour postprandial glucose >200 persisting for more than 48 hours. It is believed that national screening guidelines are unfortunately not being met, particularly while inpatient. At our institution, there are 137 pediatric patients with CF; of those, 8 have a diagnosis of CFRD, and 4 have impaired glucose tolerance.

We aim to study the adherence of our institution to the best practice guidelines for CFRD screening in pediatric patients with Cystic Fibrosis.

Retrospective chart review is occurring through our institution's EMR for inpatient data, and through a CF database (PortCF) for outpatient data. Inclusion criteria includes pediatric patients (below 1 day or above 17 years and 364 days) with CF. Exclusion criteria is those outside this age range, and those with CFRD. Consent is waived, as this is a retrospective data collection. Several variables including demographics, glycemic status, CFTR modulator and class, corticosteroid and vitamin use, and feeding regimen are also being reviewed. REDCap is being used for secure data entry and analysis. Descriptive statistical analysis will be used. Categorical data will be expressed